normal nutrition of nerve tissues. It was further shown that staleness of rice, or the development in it of poisonous substances subsequent to its being milled are not important factors. Parboiled rice, in itself healthy, when extracted with alcohol, caused polyneuritis in fowls, but the addition of the alcoholic extract to a rice known to be injurious prevented this disease. Further chemical investigations showed that the power of a rice to produce polyneuritis gallinarum varied with its phosphorus content—the higher the phosphorus content, the less liable was it to be injurious. The highest percentage of phosphorus was found to be present in rice polishings. Moreover, the addition to an injurious rice of a quantity of polishings which contained enough phosphorus to bring the total phosphorus content up to that of parboiled rice sufficed to preserve nutritive equilibrium. The prevention of beri-beri thus depends on the substitution of ordinary white rice, by a rice in which the polishing process has been omitted, or carried out to a minimal extent, or by the addition to a white rice diet of articles rich in those substances which are not present in sufficient amount in white rice. One such article, which is cheap, and may be readily obtained, is the polishings from white rice.

The Physiology of the Immediate Reaction of Anaphylaxis.—On the injection of a dose of horse serum into the vein of a previously highly sensitive guinea-pig, there occurs a chain of symptoms—chiefly of respiratory nature, which result in the death of the animal in from three to five minutes. While the general type of the reaction has been reported and confirmed by various observers, it has remained for AUER and LEWIS (*Jour. Exper. Med.*, 1910, xii, 151) to study and explain the physiological basis. While convulsive and paralytic symptoms may dominate the picture if the animal is loose, they found that if it is held in a suitable holder these are less marked and the respiratory changes come into the foreground. They thus paid especial attention to the lungs, and found, as did Gay and Southard, that at autopsy the lung in acute anaphylaxis tends to remain in an inspiratory, distended condition with open thorax, with unobstructed trachea, and large bronchi, and without obvious pulmonary œdema. This immobilization of the lungs they consider to be the most characteristic sign of immediate anaphylaxis in the guinea-pig. Experiments were performed in which the respiratory movements of the chest and the volume changes of the pleural cavity were recorded, as well as others in which the animals were allowed to breathe from a bottle, and changes during inspiration and expiration registered. As a result it was evident that some stenosis is gradually produced in the pulmonary passages so that in the final stage practically no air enters or leaves the lung in spite of violent respiratory attempts. Death is due to asphyxia. The characteristic reaction of anaphylaxis was also obtained in pithed animals, showing that its production depends on a peripheral process in the lung, and not on the central nervous system. After reviewing the possible cause of the condition, the authors conclude that it is due to a tetanic contraction of the muscles of the finer bronchioles, so that air is imprisoned in the areolar sacs. Atropine, which paralyzes the bronchial muscles, may, under certain conditions, be able to relax the anaphylactic lung so that it is again able to expand and contract. The blood pressure in immediate anaphylaxis first shows a considerable rise, and then a gradual drop to 10 to 20 mm. Shortly after the injection of the toxic dose, a heart block, often with a 3 to 1 rhythm, develops, and is probably due to asphyxia. The cardiac vagus gradually loses its irritability after injection of the toxic dose.

Jaundice in Pneumonia.—As a result of the experimental study of cholecystitis, LEMIERRE and ABRAMI have previously shown the important part played by descending, hematogeneous infections in the production of inflammation of the bile passages. They now (*La presse méd.*, 1910, No. 10, 82) report three fatal cases of pneumonia associated with jaundice in which bacteriological examination of the bile at autopsy showed pure cultures of pneumococcus. In all three instances the stools were colorless, but careful search revealed no obstruction in the bile passages. The fluid in the gall-bladder was nearly colorless, and in two of the cases failed to give a Gmelin reaction. They believe that the primary cause of the jaundice is an involvement of the liver parenchyma, and that any inflammation of the bile ducts is purely secondary. In all of the cases there were signs of alcoholic cirrhosis of the liver, and Lemierre and Abrami consider that hepatitis complicating pneumonia is rare except when the liver has been the seat of some previous pathological process.

On the Quantity of Glycuronic Acid in the Urine in Health and Disease.—TOLLENS and STERN (*Hoppe-Seyler's Ztschr. f. physiol. Chemie*, 1910, lxiv, 39) have found, by means of a new quantitative method recently described by Tollens, that the excretion of glycuronic acid in the urine is far greater than has generally been supposed. Mayer and Neuberg, for example, give the daily average output as 0.004 gm. per 100 c.c., whereas the authors find 0.025 gm. per 100 c.c. or 0.3 to 0.4 gm. in the twenty-four hours' urine as the average in health. In several cases of diabetic coma, they have encountered a complete absence of glycuronates in the urine, tested with the naphthoresorcin test. Administration of sodium salicylate, which causes a marked augmentation of the glycuronates in the urine as a rule, failed to produce a positive reaction in these cases. Various drugs, especially salicylates and chloral hydrate, combine with glycuronic acid in the body; after their administration, the glycuronic acid may amount to 1.4 gm. per diem. In one case of carbolic acid poisoning (25 gm.? taken) the urine, blackish green in color and definitely levorotatory, contained 8.5 gm. of glycuronic acid on the first day. Such a urine, of course, is capable of reducing Fehling's solution.

The Cultivation of the Organism of Infantile Paralysis.—It is of interest in relation to the recent publications of Flexner and Lewis on the etiological factor of anterior poliomyelitis, to receive the results reported by LEVADITI (*Presse médicale*, 1910, No. 6, 44) in a preliminary note from the Pasteur Institute. In several experiments he has inoculated bouillon to which the blood serum of monkeys and rabbits has been added, with active filtrates containing the specific organism. In one instance the medium became cloudy after being kept in the themostat for ten days. At the end of fifteen days the culture was injected into a monkey. After